Cell Membrane Injury In Barley (Hordeum Vulgare L.) Associated With Infection By Drechslera Graminea, Stripe Disease

Mredula Trivedi, Archana Singh

Abstract: Cell membrane injury was observed in naturally infected (weakly, moderately and heavily) and artificially inoculated seedlings of barley at different time of infection. The cell membrane injury increased with increased time of infection in both naturally infected and artificially inoculated seedlings by Drechslera graminea.

Keywords: Barley, Cell membrane injury, Drechslera graminea and Stripe disease

1 INTRODUCTION

Barley (Hordeum vulgare L.), a member of the grass family, is a major cereal grain. H. vulgare, the fourth important world crop, used for animal feed, beer, and human food was domesticated polyphyletically by humans 10,000 years ago in the Neolithic revolution in at least three centers [1]. Stripe disease is a major disease in our country causing losses as high as 70 to 72 per cent under epiphytotic conditions [2]. Drechslera graminea (Rabenh. ex Schlecht.) Shoemaker (sexual Pyrenophora graminea) is the causal agent of barley stripe. Barley stripe is disease of barley that once caused significant crop yield losses in many areas of the world. Alterations in plant water relations due to pathogenic interference has been known since long [3] . Cell wall defense structures involve morphological changes invaded by the pathogen. The outer layer of cell wall of epidermal cells in contact with incompatible pathogen swells and produces an anamorphous and fibrillar material that surrounds and trap the pathogen and prevents them from multiplying. Cell wall thickness in response to several pathogens by producing a type of cellulosic material. This material however is often infused with phenolic substances that are cross linked and further increase its resistance to penetration. Thus during the infection process a series of structural features appear on the host cell wall and the tissues, biochemical and molecular reactions take place with exchange of series of signals, messages and dialogues between host and the pathogen [4].

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2 MATERIAL AND METHOD

Raising of crop: The crop was raised in earthen pots (height 30 cm, diameter 20 cm) filled with sterile coarse sand (pH 8.3). Two barley samples Br32 and Br59 (naturally infected) and one barley sample Br70 (artificially inoculated) were taken for conducting studies.

Artificial inoculation: Artificial inoculation of Drechslera graminea was made in barley seeds of sample Br70. Naturally infected barley seeds were used for the isolation of test pathogen Drechslera graminea. Their pure culture were raised on PDA medium. For inoculation, seeds from healthy lots were surface sterilized with 1% aqueous solution of sodium hypochlorite and soaked in suspension (1×10^5 spores/ml) from 15 days old sporulating cultures of the pathogen for 24 h.

Estimation of cell membrane injury: Seedlings of healthy, naturally infected (weakly, moderately and heavily) and artificially inoculated barley plants were taken for following physiological studies at different time of infection. Cell membrane injury was estimated by the method described by Sullivan [5]. Six leaves per replication of barley categorized healthy, naturally infected (weakly, moderately and heavily) with Drechslera graminea and artificially inoculated were taken for detecting cell membrane injury. Electrical Conductance (EC) was measured by conductivity bridge of normal and autoclaved tissues. The percent injury of tissue was calculated by the following formula.

% Injury in normal tissue = Conductivity of the tissue / Total conductivity × 100

% Injury in stressed tissue = Conductivity of the tissue / Total conductivity × 100

% uninjured tissue = 100 - % injury

% Membrane injury = 100 – [(% injured stressed tissue) / (% uninjured normal tissue) × 100]

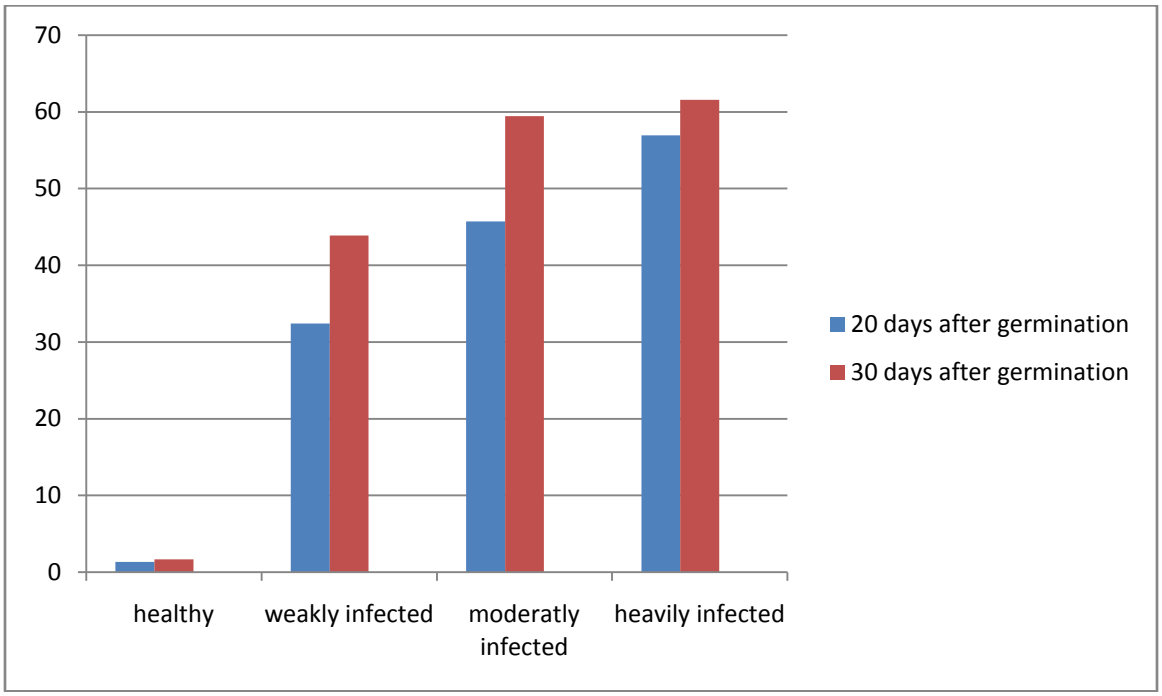
3 RESULT AND DISCUSSION

The cell membrane injury increased with increased days of infection. The changes were very much evident in both samples Br32 and Br59 of naturally infected by Drechslera graminea as well as in artificially inoculated sample Br70. It was higher at 30 days of infection in both naturally infected and artificially inoculated seedlings of barley. The cell membrane injury was highest in heavily infected seedling of

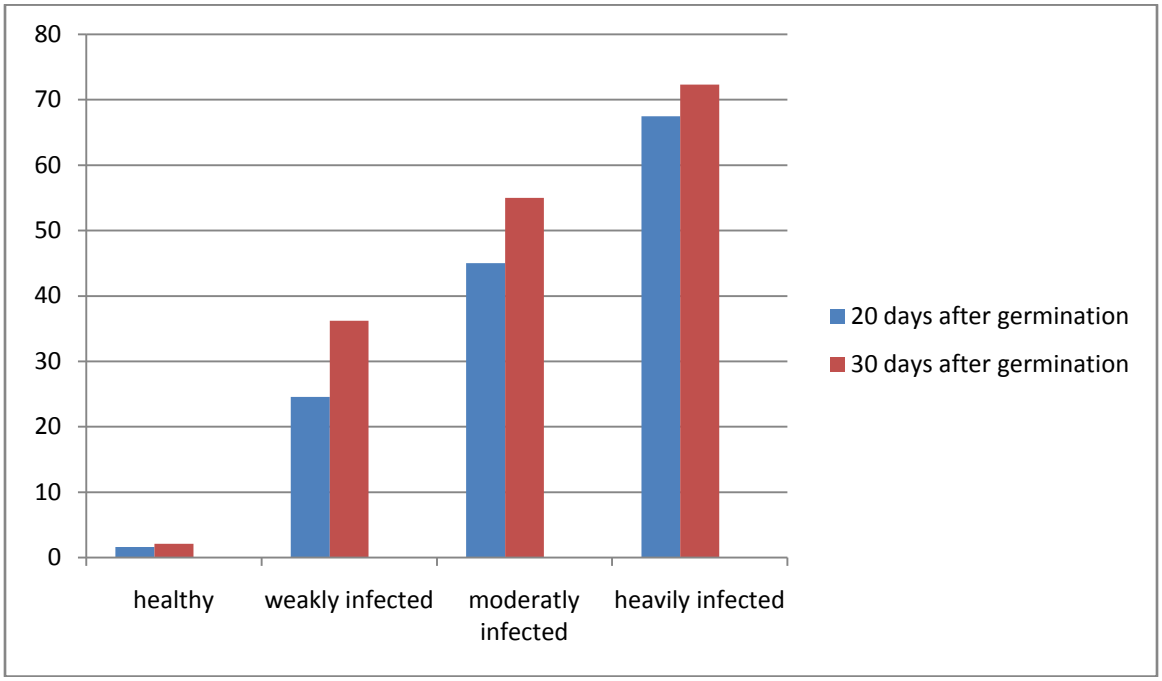
naturally infected. An increase in cell membrane injury was associated with disease development. The results indicate that increased Electrolyte leakages are one of the initial response of barley to infection by Drechslera graminea. The increase in cell membrane injury in heavily infected seedlings may be due to different physiological interactions. The plant cell wall acts as a barrier to penetration by fungal pathogens. These include the secretion of a range of plant cell wall degrading enzymes (Depolymerases) and the production of the fungal toxins such as oxalic acids by fungal pathogens. Schillerg et al also supported that developed strategy that could have potential to reduce pathogen infection is immunomodulation, the expression of genes encoding antibodies or plant bodies that could bind to pathogen virulence products [6] Belanger and Bushnell supported in their study that Fungi typically initiate an invasion of plant tissues by penetrating the plant cell wall. If the intruding pathogen can be intercepted at this early stage, cellular integrity and homeostasis are maintained, and damage to plant tissues is greatly reduced [7]. In general, The integrity of plasma membrane is a very important factor for host survival. Irreversible membrane damage in infected seedlings, is a crucial point in cell metabolism and is directly associated with hyper sensitive response also reported by heath [8]. Changes occur in membrane permeability in early disease process and therefore, could affect the development of the pathogen at this crucial stage of pathogenesis in both naturally infected and artificially inoculated seedlings. The impairment of membrane permeability would greatly influence the normal physiological functioning of the host cells [9].

4 CONCLUSION

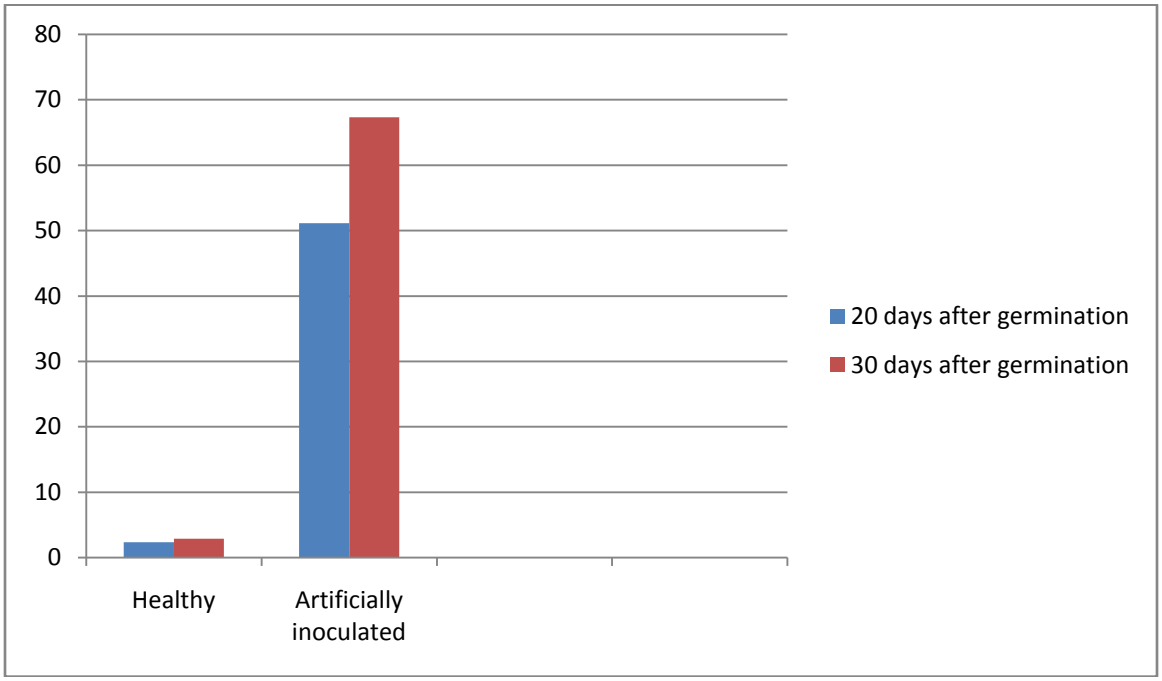
Our findings further agree with the fact that the permeability properties of cell membranes are fundamental to normal functioning of the cell.



% Cell Membrane Injury in Naturally Infected seedlings Sample Br32



% Cell Membrane Injury in Naturally Infected seedling of Sample Br59



% Cell Membrane Injury in Artificially Inoculated seedlings of Sample Br70

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